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Case Report

Primary aldosteronism associated with a giant coronary aneurysm after drug-eluting stent implantation

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ABSTRACT

Although primary aldosteronism had been recognized to be a treatable type of hypertension, it was recently suggested to be associated with an increased risk of cardiovascular complications. Coronary artery aneurysm is a rare complication after drug-eluting stent (DES) implantation, and a giant coronary aneurysm is very rare. The present case is a 51-year-old, hypertensive patient with primary aldosteronism who developed myocardial infarction, a giant coronary aneurysm after DES implantation, and then cerebral hemorrhage. Our case suggests the excessively high risk for cardiovascular complications in patients with primary aldosteronism.

<**Learning objective:** Primary aldosteronism had been recognized to be a treatable type of hypertension. However, recent studies suggest that primay aldosteronism is associated with an increased risk of cardiovascular complications rather than essential hypertension. Coronary artery aneurysm is a rare complication after DES implantation. The present case is a patient with primary aldosteronism who developed myocardial infarction, a giant coronary aneurysm after DES implantation, and then cerebral hemorrhage, thus suggesting the excessively high risk for cardiovascular complications in primary aldosteronism.>

Introduction

Primary aldosteronism had been recognized to be a treatable type of hypertension. However, recent studies suggest that primary aldosteronism is associated with an increased risk of cardiovascular complications rather than essential hypertension [1,2]. The present case is a patient with primary aldosteronism who developed myocardial infarction (MI), a giant coronary aneurysm after drug-eluting stent (DES) implantation, and then cerebral hemorrhage, thus suggesting the high risk for cardiovascular complications in primary aldosteronism.

Case report

The present case was a 51-year-old man who had been hypertensive since he was 35 years of age and he had been taking 40 mg/day nifedipine, 80 mg/day valsartan, and 50 mg/day spironolactone. He was referred to our hospital because of suspected secondary hypertension for hypokalemia (2.9 mmol/dl) at 45 years of age. However, plasma renin and aldosterone levels were 2.6 ng/ml/h and 25.1 pg/ml, respectively, and abdominal ultrasound sonography did not detect any adrenal mass or renal artery stenosis. He suddenly experienced chest pain for the first time at 47 years of age. He had no coronary risk factor except for hypertension, but his blood pressures had been around 130/80 mmHg on medication. He had no history of smoking, and his total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and hemoglobin A1c levels were 190 mg/dl, 122 mg/dl, 46 mg/dl, and 5.4%, respectively. His electrocardiogram showed ST-segment elevation in leads II, III, and aVF. He was diagnosed to have acute inferior MI. Emergent coronary angiography revealed total occlusion at the middle site of the right coronary artery (RCA) and severe narrowings at the proximal site of the left anterior descending artery (LAD) and at the first diagonal branch (D1). Primary percutaneous coronary intervention (PCI) with a bare metal stent (4.0/23 mm, Multi-Link Vision™, Abbott, Abott Park, IL, USA) implantation was performed for the RCA occlusion. Elective PCI to the LAD and D1 narrowings was also performed 1 week later with sirolimus-eluting stents (3.0/33 mm and 3.0/18 mm, Cypher™, Cordis, Miami Lakes, FL,

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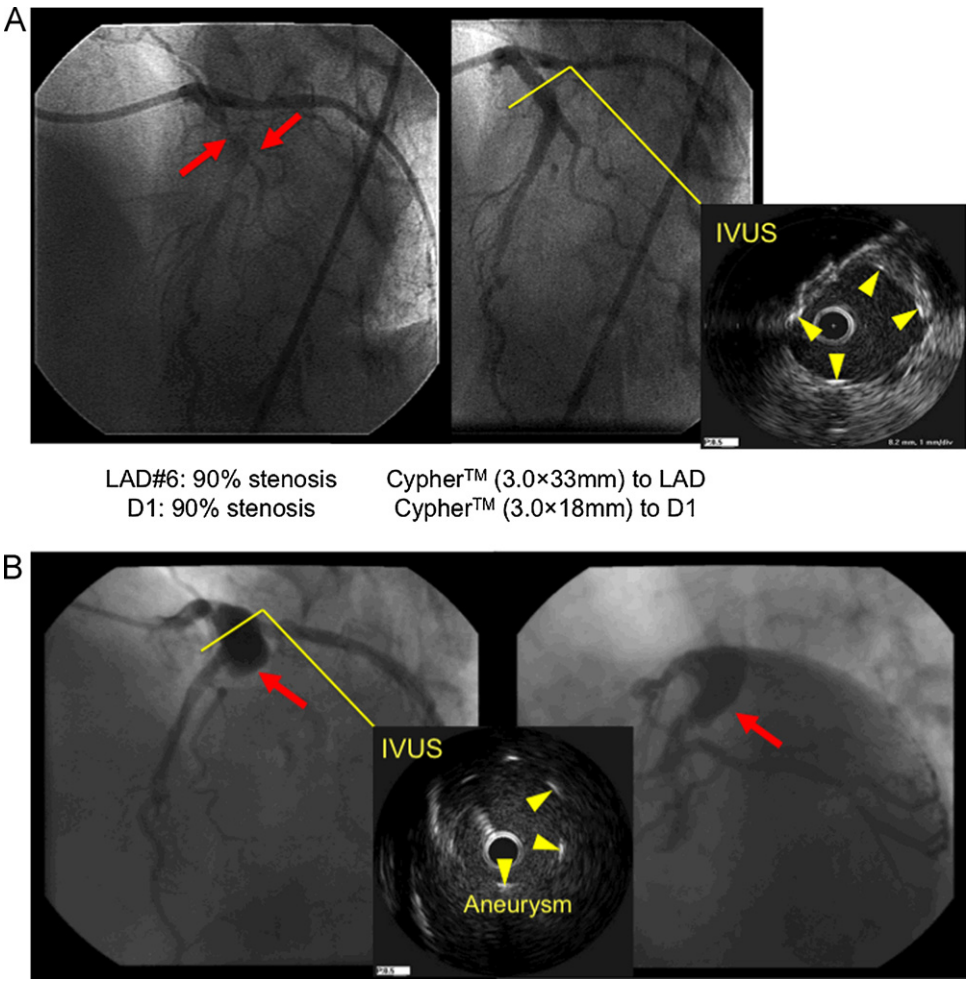

Fig. 1. Coronary angiography and intravascular ultrasound (IVUS). (A) Coronary angiography revealed severe narrowings (arrows) at the proximal site of the left anterior descending artery (LAD) and at the first diagonal branch (D1) (left panel). Percutaneous coronary intervention to the LAD and D1 narrowings was performed with sirolimus-eluting stents (Cypher™) implantation (right panel). IVUS revealed complete stent strut apposition at the stented site of the LAD. Arrow heads indicate stent strut. (B) Three years after acute myocardial infarction, coronary angiography revealed giant coronary artery aneurysm formation (arrows) at the stented site of the LAD with total occlusion of the D1. IVUS revealed stent malapposition and a giant aneurysm formation at the stented site of the LAD. Arrow heads indicate stent strut.

USA) implantation (Fig. 1A). Complete stent strut apposition was confirmed by intravascular ultrasound (IVUS) (Fig. 1A). He was discharged on dual antiplatelet therapy and 10 mg/day atorvastatin without any in-hospital events. His blood pressures were around 130/80 mmHg on anti-hypertensive drugs (10 mg/day carvedilol, 40 mg/day nifedipine, 80 mg/day valsartan, and 25 mg/day spironolactone).

He presented to the emergency room with chest pain again at 50 years of age. Although his electrocardiogram showed no ST-segment elevation, emergent coronary angiography revealed a giant coronary aneurysm formation at the stented site of the LAD with the total occlusion of the D1 (Fig. 1B). IVUS also revealed stent malapposition and a giant aneurysm formation at the stented site of the LAD (Fig. 1B). However, no aneurysm formation was found at the stented site of the RCA. PCI for the D1 occlusion failed to achieve reperfusion. Three days later, coronary computed tomography (CT) demonstrated a giant coronary artery aneurysm (19 mm in diameter, 29 mm in length) with stent malapposition and thrombus at the stented site of the LAD (Fig. 2). Anticoagulation therapy with warfarin was started to prevent thrombus formation within coronary aneurysm, and the international normalized ratio (INR) was around 2.0. Three months later, CT showed no change in the size of coronary artery aneurysm.

He complained of sudden headache, gait disturbance, and dysarthria at 51 years of age. He was diagnosed to have brain stem hemorrhage by brain CT, but the INR was 1.5. At the time of this admission, plasma levels of low renin (0.3 ng/ml/h) and high aldosterone (436.0 pg/ml) suggested primary aldosteronism. Both furosemide provocation and captril challenge tests were positive. Abdominal CT demonstrated a right adrenal mass (13 mm × 9 mm), which also showed abnormally increased uptake of ^{131}I-adosterol on adrenal scintigrams (Fig. 3). He underwent right adrenalectomy, and he was finally diagnosed as having primary aldosteronism due to adrenal adenoma.

Discussion

Although primary aldosteronism had been recognized as a treatable type of hypertension, it was recently suggested to be associated with increased risk of cardiovascular complications [1,2]. Milliez et al. investigated cardiovascular events in 124 patients with primary aldosteronism and 465 with essential hypertension [1]. They reported the prevalence of stroke and MI to be higher in primary aldosteronism than in essential hypertension. Catena et al. also showed the prevalence of cardiovascular events to be higher in 54 patients with primary aldosteronism than in 323

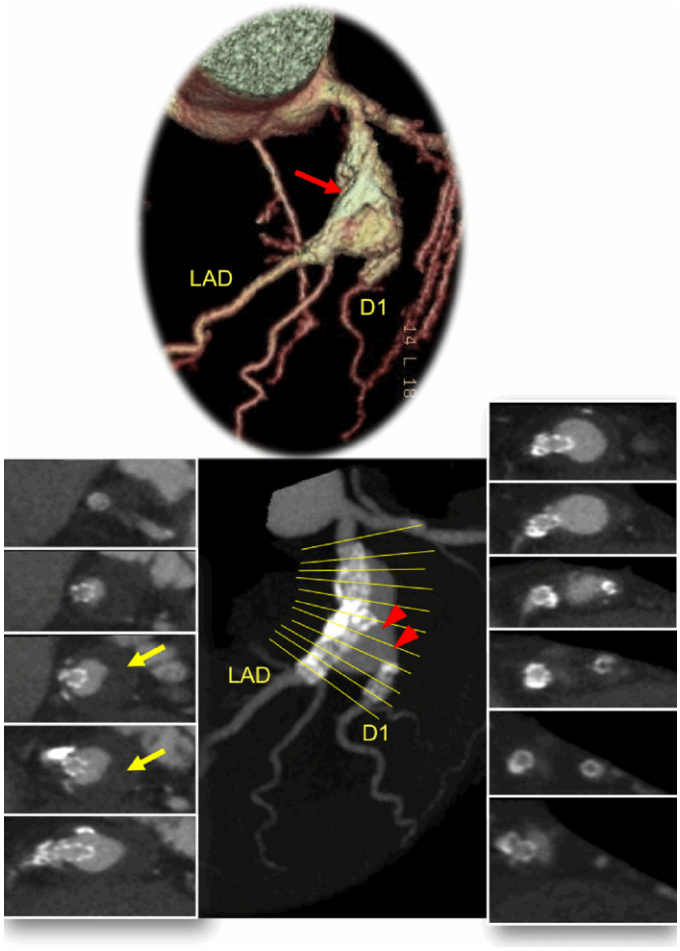

Fig. 2. The volume-rendered (upper panel) and curved and axial multiplanar reformation images (lower panels) of coronary computed tomography (CT). Coronary CT demonstrated a giant coronary aneurysm (19 mm in diameter, 29 mm in length) (red arrow) with stent malapposition and thrombus (yellow arrow) at the stented site of left anterior descending artery (LAD). CT also showed the reopened D1 as well as the stent fracture at D1 (arrow head), suggesting that D1 occlusion would be caused by stent fracture and giant coronary aneurysm.

with essential hypertension [2]. Excess aldosterone levels were reported to induce oxidative stress, inflammation, endothelial dysfunction, and increased arterial stiffness [3–6]. The current case with primary aldosteronism developed MI, a giant coronary aneurysm after DES implantation, and then cerebral hemorrhage. These findings and the current case suggest the excessively high risk for cardiovascular complications in primary aldosteronism.

Coronary aneurysms were reported to be a rare complication after DES implantation (1%), and a giant aneurysm (>8 mm in diameter) is much rarer [7,8]. Anandaraja et al. reported a giant coronary aneurysm (8 mm × 35 mm) after DES (TAXUS™, Boston Scientific, Natick, MA, USA) implantation [9]. Okamura et al. also reported a giant coronary aneurysm (8 mm × 17 mm) after DES (Cypher™) implantation [10]. The mechanism for coronary aneurysm formation after DES implantation remains unknown, but extensive acute vessel damage during the PCI procedure, hypersensitivity reactions to DES, infectious processes, and late-acquired malapposition with excessive vessel remodeling may be implicated [7,8]. The current case with primary aldosteronism developed a giant coronary artery aneurysm (19 mm × 29 mm) after DES implantation. Although there has been no reported case with coronary aneurysm associated with primary aldosteronism, excessive oxidative stress and inflammation in primary aldosteronism may have contributed to this giant coronary aneurysm formation [4–6]. Since coronary aneurysms with a diameter of >30 mm are suggested to be at high risk for rupture, such aneurysms should be surgically treated [11]. Moreover, no ruptured coronary aneurysm after DES implantation has been reported. Therefore, our case was followed medically without surgical resection. However, since the prognosis of coronary aneurysms after DES implantation remains unknown, we will carefully follow this case.

Primary aldosteronism is associated with increased cardiovascular complications, and it can be effectively treated with adrenalectomy [2], thus it is important to diagnose and treat primary aldosteronism in a timely manner for hypertensive patients.

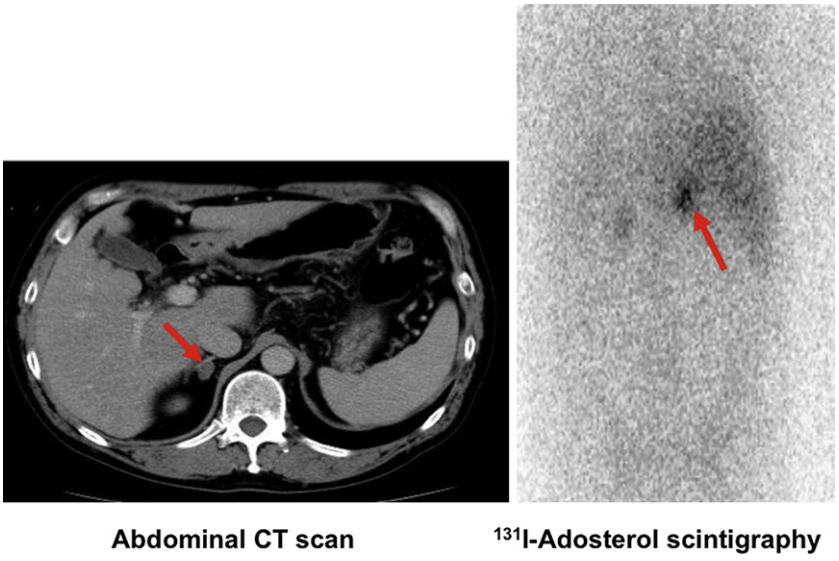

Fig. 3. Abdominal computed tomography (CT) and ^{131}I-adosterol scintigraphy. Abdominal CT demonstrated a right adrenal mass (13 mm × 9 mm) (left panel), which also had abnormally increased uptake of ^{131}I-adosterol on adrenal scintigrams (right panel). Arrows indicate adrenal mass.

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